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SESSION RESUMED IN FILE 'HOME' AT 10:34:45 ON 10 JUN 2009

FILE 'HOME' ENTERED AT 10:34:45 ON 10 JUN 2009

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.20	2.20

=> file medline biosis caplus embase

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.64	2.64

FILE 'MEDLINE' ENTERED AT 10:36:03 ON 10 JUN 2009

FILE 'BIOSIS' ENTERED AT 10:36:03 ON 10 JUN 2009

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FILE 'CAPLUS' ENTERED AT 10:36:03 ON 10 JUN 2009

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FILE 'EMBASE' ENTERED AT 10:36:03 ON 10 JUN 2009

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=> s periodontal

L1 87167 PERIODONTAL

=> s l1 and neurotroph?

L2 100 L1 AND NEUROTROPH?

=> s l2 (implant or transplant)

MISSING OPERATOR 'L10 (IMPLANT'

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s l2 and (implant or transplant)

L3 10 L2 AND (IMPLANT OR TRANSPLANT)

=> dup rem l2

PROCESSING COMPLETED FOR L2

L4 41 DUP REM L2 (59 DUPLICATES REMOVED)

=> dup rem l3

PROCESSING COMPLETED FOR L3

L5 7 DUP REM L3 (3 DUPLICATES REMOVED)

=> dis his

(FILE 'HOME' ENTERED AT 10:26:43 ON 10 JUN 2009)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:36:03 ON 10 JUN 2009

L1 87167 S PERIODONTAL
L2 100 S L1 AND NEUROTROPH?
L3 10 S L2 AND (IMPLANT OR TRANSPLANT)
L4 41 DUP REM L2 (59 DUPLICATES REMOVED)
L5 7 DUP REM L3 (3 DUPLICATES REMOVED)

=> dis ibib abs l5 1-7

L5 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:1529891 CAPLUS

DOCUMENT NUMBER: 150:71207

TITLE: Treatment of diseases and disorders using
self-renewing colony forming cells cultured and
expanded in vitro

INVENTOR(S): Kopen, Gene; Wagner, Joseph; Ragaglia, Vanessa;
Heimbach, Baron; Gore, Richard S.

PATENT ASSIGNEE(S): Neuronyx, Inc., USA

SOURCE: PCT Int. Appl., 138pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008156728	A1	20081224	WO 2008-US7488	20080616
W:	AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
US 20090053183	A1	20090226	US 2008-140065	20080616
PRIORITY APPLN. INFO.:			US 2007-929151P	P 20070615
			US 2007-929152P	P 20070615
			US 2007-955204P	P 20070810
			US 2007-996093P	P 20071101
AB	The present invention relates to methods and uses of cells for the prevention and treatment of a wide variety of diseases and disorders and the repair and regeneration of tissues and organs using low passage and extensively passaged in vitro cultured, self-renewing, colony forming somatic cells (CF-SC). For example, adult bone marrow-derived somatic cells (ABM-SC), or compns. produced by such cells, are useful alone or in combination with other components for treating, for example, cardiovascular, neurol., integumentary, dermatol., periodontal, and immune mediated diseases, disorders, pathologies, and injuries.			
REFERENCE COUNT:	5	THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT		

L5 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:285972 CAPLUS

DOCUMENT NUMBER: 148:315434

TITLE: Calcium phosphate nanofibers

INVENTOR(S): Tan, Jian; Joo, Yong L.

PATENT ASSIGNEE(S): Cornell Research Foundation, Inc., USA
 SOURCE: PCT Int. Appl., 43pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008028194	A2	20080306	WO 2007-US77560	20070904
WO 2008028194	A3	20081204		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			

PRIORITY APPLN. INFO.: US 2006-824377P P 20060901

AB This invention relates to calcium-phosphate nanofiber matrixes comprising randomly dispersed crystalline calcium-phosphate nanofibers. The nanofibers are synthesized using sol-gel methods combined with electrospinning. The nanofibers may be hollow, solid or may comprise a calcium-phosphate shell surrounding a polymer containing inner core to which biol. functional additives may be added. The nanofiber matrixes may be used to culture bone and dental cells, and as implants to treat bone, dental or periodontal diseases and defects.

L5 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:1161563 CAPLUS

DOCUMENT NUMBER: 150:71245

TITLE: Clinical outcomes with bioactive agents alone or in combination with grafting or guided tissue regeneration

AUTHOR(S): Trombelli, Leonardo; Farina, Roberto

CORPORATE SOURCE: Research Centre for the Study of Periodontal Diseases, University of Ferrara, Ferrara, Italy

SOURCE: Journal of Clinical Periodontology (2008), 35(Suppl. 8), 117-135

CODEN: JCPEDZ; ISSN: 0303-6979

PUBLISHER: Wiley-Blackwell

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. The purpose of the present review was to determine the clin. effect of the use of bioactive agents (BAs) for the treatment of intra-osseous and furcation defects. The effectiveness of the BAs was evaluated when used in addition to open flap debridement either alone or in association with grafts and/or guided tissue regeneration (GTR). Among the included agents, recombinant human platelet-derived growth factor-BB (rhPDGF-BB), platelet-rich plasma (PRP), com. available enamel matrix derivative (cEMD) and peptide P-15 (P-15) have been clin. tested for treating periodontal defects. The results of the present review indicate that: (1) cEMD either alone or in combination with grafts can be effectively used to treat intra-osseous defects and the clin. results appear to be stable long term; (2) the addnl. use of a graft seems to enhance the clin. outcome of cEMD; (3) the combined use of rhPDGF-BB and

P-15 with a graft biomaterial has shown beneficial effects in intraosseous defects; (4) contrasting results were reported for PRP and graft combinations; and (5) limited evidence supports the use of BAs either alone or in association with graft/GTR for the treatment of furcation defects.

REFERENCE COUNT: 190 THERE ARE 190 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2007485083 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17379425
TITLE: Schwann cell graft: a method to promote sensory responses of osseointegrated implants.
AUTHOR: Yuan Quan; Gong Ping; Tan Zhen
CORPORATE SOURCE: Oral Implant Center, West China College of Stomatology, Sichuan University, Chengdu 610041, China.
SOURCE: Medical hypotheses, (2007) Vol. 69, No. 4, pp. 800-3. Electronic Publication: 2007-03-26. Journal code: 7505668. ISSN: 0306-9877.
PUB. COUNTRY: Scotland: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200710
ENTRY DATE: Entered STN: 21 Aug 2007
Last Updated on STN: 25 Oct 2007
Entered Medline: 24 Oct 2007

AB Osseointegrated dental implants have been widely used in clinics to restore the missing teeth of patients. Since there are no periodontal ligament and associated Ruffini endings in the peri-implant tissues, sensory thresholds of the implant are much higher than those of natural teeth, and its self-protective reflex is quit poor. Implant fracture or aggressive bone loss sometimes occurs because the patient cannot feel the overloads exerted on the implant. Until now, no available method has been issued to solve such a problem. Schwann cell is the glial cell of peripheral nerve system. It has been widely accepted to play indispensable roles during neural development and regeneration. Its mechanism includes forming Bungner's band, producing neurotrophic factors, synthesizing surface cell adhesion molecules, and elaborating basement membrane. Furthermore, Schwann cell is quite important for the periodontal Ruffini endings. Applying these functions of Schwann cells, we put forward a hypothesis that transplanting Schwann cells into the implant site can be a method to promote sensory responses of the dental implants.

L5 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:635003 CAPLUS
DOCUMENT NUMBER: 145:90170
TITLE: Tissue engineering devices for the repair and regeneration of tissue
INVENTOR(S): Seyda, Agnieszka; Colter, David C.; Buensuceso, Charito S.; Sridevi, Dhanaraj; Gosiewska, Anna; Geesin, Jeffrey C.; Scopelianos, Angelo G.
PATENT ASSIGNEE(S): Ethicon, Inc., USA
SOURCE: PCT Int. Appl., 32 pp. CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006068972	A2	20060629	WO 2005-US45732	20051215
WO 2006068972	A3	20070503		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GO, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			
US 20060171930	A1	20060803	US 2005-297156	20051208
AU 2005319401	A1	20060629	AU 2005-319401	20051215
CA 2591979	A1	20060629	CA 2005-2591979	20051215
EP 1835949	A2	20070926	EP 2005-854448	20051215
R:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU			
JP 2008523957	T	20080710	JP 2007-548345	20051215
PRIORITY APPLN. INFO.:			US 2004-637984P	P 20041221
			WO 2005-US45732	W 20051215

AB Tissue engineering devices for use in the repair or regeneration of tissue made of support scaffolds and cell sheets. The seeding of cells on scaffolds using cell sheets was compared against the conventional method of seeding scaffolds with cell suspension. Seeding of a biodegradable, biocompatible scaffold with cell sheets provides equivalent cell viability and cell distribution to seeding by cell suspension. Therefore, seeding scaffolds using the cell sheet method is a good alternative to the conventional method of cell suspension seeding with the benefit of one-step application and more controlled device handling.

L5 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:681122 CAPLUS
DOCUMENT NUMBER: 145:130940
TITLE: Tissue engineering devices with polymeric support scaffolds for the repair and regeneration of tissue
INVENTOR(S): Seyda, Agnieszka; Colter, David C.; Buensuceso, Charito S.; Dhanaraj, Sridevi; Gosiewska, Anna; Geesin, Jeffrey C.; Scopelianos, Angelo
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 12 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20060153815	A1	20060713	US 2005-304091	20051215
PRIORITY APPLN. INFO.:			US 2004-637781P	P 20041221
AB	Tissue engineering devices for use in the repair or regeneration of tissue made of support scaffolds and cell sheets are described. Thus, cells were subjected to cell sheet preparation and were combined with nonwoven, degradable scaffolds as a first step towards generation of tissue engineering devices. Osteoblasts/chondrocytes for musculoskeletal applications as well as urothelial cells/bladder smooth muscle cells for urogenital			

applications were tested. Cells were cultured prior to scaffold deposition, e.g., in sheets on thermoresponsive poly(NIPAAm)-coated dishes.

L5 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2009 ACS ON STN

ACCESSION NUMBER: 2005:25902 CAPLUS

DOCUMENT NUMBER: 142:303690

TITLE: Remedy and therapeutic method for periodontal diseases and pulpal diseases with neurotrophic factors

INVENTOR(S): Kurihara, Hidemi; Kawaguchi, Hiroyuki; Takeda, Katsuhiko; Shiba, Hideki; Mizuno, Noriyoshi; Yoshino, Hiroshi; Hasegawa, Naohiko; Shinohara, Hiroaki

PATENT ASSIGNEE(S): Two Cells Co. Ltd., Japan

SOURCE: PCT Int. Appl., 86 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005025605	A1	20050324	WO 2004-JP13023	20040908
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004271843	A1	20050324	AU 2004-271843	20040908
EP 1671641	A1	20060621	EP 2004-787706	20040908
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
CN 1871024	A	20061129	CN 2004-80031194	20040908
RU 2336089	C2	20081020	RU 2006-111465	20040908
US 20070071693	A1	20070329	US 2006-571069	20061207
PRIORITY APPLN. INFO.:			JP 2003-316719	A 20030909
			WO 2004-JP13023	W 20040908

AB It is intended to provide a remedy and a therapeutic method for periodontal diseases and pulpal diseases, a transplantation material for regenerating a periodontal tissue and a method of regenerating a periodontal tissue. Namely, a remedy for periodontal diseases and pulpal diseases comprising a neurotrophic factor as the active ingredient. The effect of brain-derived neurotrophic factor (BDNF) on cultured human periodontal ligament cell and human gingival keratinocyte was examined

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> dis his

(FILE 'HOME' ENTERED AT 10:26:43 ON 10 JUN 2009)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:36:03 ON 10 JUN 2009

L1 87167 S PERIODONTAL
 L2 100 S L1 AND NEUROTROPH?
 L3 10 S L2 AND (IMPLANT OR TRANSPLANT)
 L4 41 DUP REM L2 (59 DUPLICATES REMOVED)
 L5 7 DUP REM L3 (3 DUPLICATES REMOVED)

=> dis ibib abs l4 1-41

L4 ANSWER 1 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
 ACCESSION NUMBER: 2008:1529891 CAPLUS
 DOCUMENT NUMBER: 150:71207
 TITLE: Treatment of diseases and disorders using
 self-renewing colony forming cells cultured and
 expanded in vitro
 INVENTOR(S): Kopen, Gene; Wagner, Joseph; Ragaglia, Vanessa;
 Heimbach, Baron; Gore, Richard S.
 PATENT ASSIGNEE(S): Neuronyx, Inc., USA
 SOURCE: PCI Int. Appl., 138pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008156728	A1	20081224	WO 2008-US7488	20080616
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 20090053183	A1	20090226	US 2008-140065	20080616
PRIORITY APPLN. INFO.:			US 2007-929151P	P 20070615
			US 2007-929152P	P 20070615
			US 2007-955204P	P 20070810
			US 2007-996093P	P 20071101
AB	The present invention relates to methods and uses of cells for the prevention and treatment of a wide variety of diseases and disorders and the repair and regeneration of tissues and organs using low passage and extensively passaged in vitro cultured, self-renewing, colony forming somatic cells (CF-SC). For example, adult bone marrow-derived somatic cells (ABM-SC), or compns. produced by such cells, are useful alone or in combination with other components for treating, for example, cardiovascular, neurol., integumentary, dermatol., periodontal, and immune mediated diseases, disorders, pathologies, and injuries.			
REFERENCE COUNT:	5	THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT		

L4 ANSWER 2 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
 ACCESSION NUMBER: 2008:285972 CAPLUS
 DOCUMENT NUMBER: 148:315434
 TITLE: Calcium phosphate nanofibers
 INVENTOR(S): Tan, Jian; Joo, Yong L.
 PATENT ASSIGNEE(S): Cornell Research Foundation, Inc., USA

SOURCE: PCT Int. Appl., 43pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008028194	A2	20080306	WO 2007-US77560	20070904
WO 2008028194	A3	20081204		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			

PRIORITY APPLN. INFO.: US 2006-824377P P 20060901

AB This invention relates to calcium-phosphate nanofiber matrixes comprising randomly dispersed crystalline calcium-phosphate nanofibers. The nanofibers are synthesized using sol-gel methods combined with electrospinning. The nanofibers may be hollow, solid or may comprise a calcium-phosphate shell surrounding a polymer containing inner core to which biol. functional additives may be added. The nanofiber matrixes may be used to culture bone and dental cells, and as implants to treat bone, dental or periodontal diseases and defects.

L4 ANSWER 3 OF 41 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2008408883 MEDLINE

DOCUMENT NUMBER: PubMed ID: 18390540

TITLE: Brain-derived neurotrophic factor stimulates bone/cementum-related protein gene expression in cementoblasts.

AUTHOR: Kajiya Mikihiro; Shiba Hideki; Fujita Tsuyoshi; Ouhara Kazuhisa; Takeda Katsuhiko; Mizuno Noriyoshi; Kawaguchi Hiroyuki; Kitagawa Masae; Takata Takashi; Tsuji Koichiro; Kurihara Hidemi

CORPORATE SOURCE: Department of Periodontal Medicine, Hiroshima University Graduate School of Biomedical Sciences, Minami-ku, Hiroshima 34-8553, Japan.

SOURCE: The Journal of biological chemistry, (2008 Jun 6) Vol. 283, No. 23, pp. 16259-67. Electronic Publication: 2008-04-03. Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200807

ENTRY DATE: Entered STN: 27 Jun 2008

Last Updated on STN: 16 Jul 2008

Entered Medline: 15 Jul 2008

AB Brain-derived neurotrophic factor (BDNF), recognized as essential in the developing nervous system, is involved in differentiation and proliferation in non-neuronal cells, such as endothelial cells, osteoblasts, and periodontal ligament cells. We have focused on

the application of BDNF to the regeneration of periodontal tissue and indicated that BDNF promotes the regeneration of experimentally created periodontal defects. Cementoblasts form cementum, mineralized tissue, which is key to establishing a functional periodontium. The application of BDNF to the regeneration of periodontal tissue requires elucidation of the mechanism by which BDNF regulates the functions of cementoblasts. In this study, we examined how BDNF regulates the mRNA expression of bone/cementum-related proteins (alkaline phosphatase (ALP), osteopontin (OPN), and bone morphogenetic protein-2 (BMP-2)) in cultures of immortalized human cementoblast-like (HCEM) cells. BDNF elevated the mRNA levels of ALP, OPN, and BMP-2 in HCEM cells. Small interfering RNA (siRNA) for TRKB, a high affinity receptor of BDNF, siRNA for ELK-1, which is a downstream target of ERK1/2, and PD98059, an ERK inhibitor, obviated the increase in the mRNA levels. BDNF increased the levels of phosphorylated ERK1/2 and Elk-1, and the blocking of BDNF signaling by treatment with siRNA for TRKB and PD98059 suppressed the phosphorylation of ERK1/2 and Elk-1. Furthermore, BDNF increased the levels of phosphorylated c-Raf, which activates the ERK signaling pathway. These findings provide the first evidence that the TrkB-c-Raf-ERK1/2-Elk-1 signaling pathway is required for the BDNF-induced mRNA expression of ALP, OPN, and BMP-2 in HCEM cells.

L4 ANSWER 4 OF 41 MEDLINE on STN DUPLICATE 2
 ACCESSION NUMBER: 2008714203 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 18980528
 TITLE: Effect of neurotrophin-4/5 on bone/cementum-related protein expressions and DNA synthesis in cultures of human periodontal ligament cells.
 AUTHOR: Mizuno Noriyoshi; Shiba Hideki; Inui Takafumi; Takeda Katsuhiko; Kajiya Mikihito; Hasegawa Naohiko; Kawaguchi Hiroyuki; Kurihara Hidemi
 CORPORATE SOURCE: Department of Periodontal Medicine, Hiroshima University Graduate School of Biomedical Sciences, Hiroshima, Japan.. mizuno@hiroshima-u.ac.jp
 SOURCE: Journal of periodontology, (2008 Nov) Vol. 79, No. 11, pp. 2182-9.
 Journal code: 8000345. ISSN: 0022-3492.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Dental Journals; Priority Journals
 ENTRY MONTH: 200902
 ENTRY DATE: Entered STN: 5 Nov 2008
 Last Updated on STN: 15 Feb 2009
 Entered Medline: 12 Feb 2009
 AB BACKGROUND: We studied neurotrophins (NTs) as signaling molecules for periodontal tissue regeneration and showed that nerve growth factor (NGF) and brain-derived neurotrophic factor (BDNF) modulate the proliferation and differentiation of human periodontal ligament (HPL) cells in vitro. The purpose of this study was to investigate whether NT-4/5 also has the ability to regulate the function of HPL cells. METHODS: mRNA expressions of NT-4/5 and its high-affinity tyrosine kinase receptor (trkB) were analyzed in HPL cells by reverse transcription-polymerase chain reaction. We examined how NT-4/5 regulates the mRNA expression of bone/cementum-related proteins (alkaline phosphatase [ALPase], osteopontin [OPN], osteocalcin [OC], and bone morphogenetic protein [BMP]-2) in cultures of HPL cells. Moreover, the effects of NT-4/5 on calcification, the production of OPN and OC, and DNA synthesis in HPL cells were examined. RESULTS: NT-4/5 and trkB mRNA were expressed in HPL cells. NT-4/5 elevated the mRNA levels of ALPase, OPN, OC, and BMP-2 and the syntheses of OPN, OC, and DNA in HPL cells.

PD98059, an extracellular signal-regulated kinase (ERK) inhibitor, obviated the increase in the mRNA levels of ALPase, OPN, OC, and BMP-2. NT-4/5 increased the levels of phosphorylated ERK1/2. Furthermore, NT-4/5 enhanced the amount of mineral deposits in cultures of HPL cells. CONCLUSION: NT-4/5, as well as BDNF and NGF, is suggested to play a role in the regulation of function of periodontal ligament cells.

L4 ANSWER 5 OF 41 MEDLINE on STN DUPLICATE 3
 ACCESSION NUMBER: 2008343797 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 18454671
 TITLE: In vitro characterization of the cytokine profile of the epithelial cell rests of Malassez.
 AUTHOR: Ohshima Mitsuhiro; Yamaguchi Yoko; Micke Patrick; Abiko Yoshimitsu; Otsuka Kichibee
 CORPORATE SOURCE: Department of Biochemistry, Nihon University School of Dentistry, 1-8-13 Kanda-Surugadai, Chiyoda-ku, Tokyo, Japan.. oshima-m@dent.nihon-u.ac.jp
 SOURCE: Journal of periodontology, (2008 May) Vol. 79, No. 5, pp. 912-9.
 Journal code: 8000345. ISSN: 0022-3492.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: (COMPARATIVE STUDY)
 (JOURNAL; ARTICLE; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Dental Journals; Priority Journals
 ENTRY MONTH: 200807
 ENTRY DATE: Entered STN: 30 May 2008
 Last Updated on STN: 3 Jul 2008
 Entered Medline: 2 Jul 2008

AB BACKGROUND: The epithelial cell rests of Malassez (ERM) are an integral part of the periodontal ligament and are considered to play an important role in dental pathology. Surprisingly, this cell type is poorly described and is often disregarded in the context of periodontal research. The aim of this study was to establish primary cell cultures of human ERM, characterize the cytokine profile, and compare it to other periodontal cell entities. METHODS: ERM-derived epithelial cells were isolated from the periodontal ligament of three subjects. A cytokine antibody array, including 120 cytokines in two membranes, was used to determine the cytokine profile of conditioned medium from the ERM-derived epithelial cells. The results were compared to those of gingival epithelial cells and periodontal ligament fibroblasts. RESULTS: ERM-derived epithelial cells expressed 29 of 120 cytokines in significant amounts, including cytokines, chemokines, growth factors, and related proteins, such as interleukin (IL)-1, -6, -8, and -10; granulocyte macrophage-colony stimulating factor; monocyte chemoattractant protein (MCP)-1, -2, and -3; amphiregulin; glial-derived neurotrophic factor; vascular endothelial growth factor; and insulin-like growth factor binding protein-2. The cytokine profile of ERM cells was similar to that of gingival epithelial cells but strikingly different from the profile of periodontal ligament fibroblasts. CONCLUSIONS: The results indicated that, via paracrine secretion of a variety of soluble factors, the ERM cells actively take part in the homeostasis of the periodontium. Therefore, future research on the pathophysiology of periodontal tissue should include this often overlooked cell type.

L4 ANSWER 6 OF 41 MEDLINE on STN DUPLICATE 4
 ACCESSION NUMBER: 2008338309 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 18446825
 TITLE: Effect of tooth loss on spatial memory and trkB-mRNA levels in rats.

AUTHOR: Yamazaki Kaoruko; Wakabayashi Noriyuki; Kobayashi Takuya;
Suzuki Tetsuya
CORPORATE SOURCE: Department of Removable Prosthodontics, School of
Dentistry, Iwate Medical University, Japan.
SOURCE: Hippocampus, (2008) Vol. 18, No. 6, pp. 542-7.
Journal code: 9108167. E-ISSN: 1098-1063.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200809
ENTRY DATE: Entered STN: 28 May 2008
Last Updated on STN: 23 Sep 2008
Entered Medline: 22 Sep 2008

AB The mechanism by which tooth loss accelerates spatial memory impairment is unknown. The purpose of this study was to test the hypothesis that tooth loss affects trkB-mRNA levels and leads to an accelerated decrease in the hippocampal cell density in rats. A radial maze was used to evaluate the spatial memory of male Wistar rats that were categorized based on the number of extracted molar teeth. Number of hippocampal pyramidal cells and the trkB-mRNA expressions in the amygdala, perirhinal cortex, thalamus, and the hippocampal CA1, CA3, and CA4 areas, were evaluated using molecular biological techniques. Seven weeks after tooth extraction, maze performance was significantly lower in each tooth loss group than in the control group, and the number of extracted teeth was inversely proportional to the induction of the trkB-mRNA and the hippocampal cell density. The average weight of rats increased by controlled feeding throughout the experiment without showing a significant difference between the control and experimental groups. The results indicated that, in rats, the spatial memory-linked trkB-mRNA was reduced in association with the tooth loss; this supports the hypothesis and suggests that teeth have a role in the prevention of spatial memory impairment.
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L4 ANSWER 7 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:1161563 CAPLUS
DOCUMENT NUMBER: 150:71245
TITLE: Clinical outcomes with bioactive agents alone or in
combination with grafting or guided tissue
regeneration
AUTHOR(S): Trombelli, Leonardo; Farina, Roberto
CORPORATE SOURCE: Research Centre for the Study of Periodontal Diseases,
University of Ferrara, Ferrara, Italy
SOURCE: Journal of Clinical Periodontology (2008), 35(Suppl.
8), 117-135
CODEN: JCPEDZ; ISSN: 0303-6979
PUBLISHER: Wiley-Blackwell
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. The purpose of the present review was to determine the clin. effect of the use of bioactive agents (BAS) for the treatment of intra-osseous and furcation defects. The effectiveness of the BAS was evaluated when used in addition to open flap debridement either alone or in association with grafts and/or guided tissue regeneration (GTR). Among the included agents, recombinant human platelet-derived growth factor-BB (rhPDGF-BB), platelet-rich plasma (PRP), com. available enamel matrix derivative (cEMD) and peptide P-15 (P-15) have been clin. tested for treating periodontal defects. The results of the present review indicate that: (1) cEMD either alone or in combination with grafts can be

effectively used to treat intra-osseous defects and the clin. results appear to be stable long term; (2) the adnl. use of a graft seems to enhance the clin. outcome of cEMD; (3) the combined use of rhPDGF-BB and P-15 with a graft biomaterial has shown beneficial effects in intraosseous defects; (4) contrasting results were reported for PRP and graft combinations; and (5) limited evidence supports the use of BAs either alone or in association with graft/GTR for the treatment of furcation defects.

REFERENCE COUNT: 190 THERE ARE 190 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 8 OF 41 MEDLINE on STN DUPLICATE 5
 ACCESSION NUMBER: 2007485083 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 17379425
 TITLE: Schwann cell graft: a method to promote sensory responses of osseointegrated implants.
 AUTHOR: Yuan Quan; Gong Ping; Tan Zhen
 CORPORATE SOURCE: Oral Implant Center, West China College of Stomatology, Sichuan University, Chengdu 610041, China.
 SOURCE: Medical hypotheses, (2007) Vol. 69, No. 4, pp. 800-3.
 Electronic Publication: 2007-03-26.
 Journal code: 7505668. ISSN: 0306-9877.
 PUB. COUNTRY: Scotland: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200710
 ENTRY DATE: Entered STN: 21 Aug 2007
 Last Updated on STN: 25 Oct 2007
 Entered Medline: 24 Oct 2007

AB Osseointegrated dental implants have been widely used in clinics to restore the missing teeth of patients. Since there are no periodontal ligament and associated Ruffini endings in the peri-implant tissues, sensory thresholds of the implant are much higher than those of natural teeth, and its self-protective reflex is quite poor. Implant fracture or aggressive bone loss sometimes occurs because the patient cannot feel the overloads exerted on the implant. Until now, no available method has been issued to solve such a problem. Schwann cell is the glial cell of peripheral nerve system. It has been widely accepted to play indispensable roles during neural development and regeneration. Its mechanism includes forming Bungner's band, producing neurotrophic factors, synthesizing surface cell adhesion molecules, and elaborating basement membrane. Furthermore, Schwann cell is quite important for the periodontal Ruffini endings. Applying these functions of Schwann cells, we put forward a hypothesis that transplanting Schwann cells into the implant site can be a method to promote sensory responses of the dental implants.

L4 ANSWER 9 OF 41 MEDLINE on STN DUPLICATE 6
 ACCESSION NUMBER: 2007056449 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 17245704
 TITLE: Involvement of neurotrophin-4/5 in regeneration of the periodontal Ruffini endings at the early stage.
 AUTHOR: Jabbar Shahiqul; Harada Fumiko; Aita Megumi; Ohishi Megumi; Saito Isao; Kawano Yoshiro; Suzuki Akiko; Nozawa-Inoue Kayoko; Maeda Takeyasu
 CORPORATE SOURCE: Division of Oral Anatomy, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan.
 SOURCE: The Journal of comparative neurology, (2007 Mar 20) Vol. 501, No. 3, pp. 400-12.
 Journal code: 0406041. ISSN: 0021-9967.

PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200703
 ENTRY DATE: Entered STN: 31 Jan 2007
 Last Updated on STN: 24 Mar 2007
 Entered Medline: 20 Mar 2007

AB Little is known about the role of neurotrophin-4/5 (NT-4/5) in the regeneration of mechanoreceptors. Therefore, the present study examined the regeneration process of Ruffini endings in the periodontal ligament in nt-4/5-deficient and wildtype mice following transection of the inferior alveolar nerve by immunohistochemistry for protein gene product 9.5 (PGP 9.5), a general neuronal marker, and by computer-assisted quantitative image analysis. Furthermore, rescue experiments by a continuous administration of recombinant NT-4/5 were performed and analyzed quantitatively. At postoperative day 3 (PO 3d), almost all PGP 9.5-positive neural elements had disappeared; they began to appear in both types of animals at PO 7d. At PO 10d, almost all nerve fibers showed a beaded appearance, with fewer ramifications in both types of mice. Although the regeneration proceeded in the wildtype, a major population of the periodontal Ruffini endings continued to display smooth outlines at PO 28d in the nt-4/5 homozygous mice. The reduction ratio of neural density reached a maximum at PO 3d, decreased at PO 10d, and later showed a plateau. In a rescue experiment, an administration of NT-4/5 showed an acceleration of nerve regeneration in the homozygous mice. These findings indicate that the nt-4/5-depletion causes a delay in the regeneration of the periodontal Ruffini endings, but the delay is shortened by an exogenous administration of NT-4/5. Combined with our previous findings of bdnf-deficient mice (Harada et al. [2003] Arch Histol Cytol 66:183-194), these morphological and numerical data suggest that multiple neurotrophins such as NT-4/5 and brain-derived neurotrophic factor (BDNF) play roles in their regeneration in a stage-specific manner.
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L4 ANSWER 10 OF 41 MEDLINE on STN DUPLICATE 7
 ACCESSION NUMBER: 2007041098 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 17178438
 TITLE: Involvement of GDNF and its receptors in the maturation of the periodontal Ruffini endings.
 AUTHOR: Igarashi Yasushi; Aita Megumi; Suzuki Akiko; Nandasena Tharanga; Kawano Yoshiro; Nozawa-Inoue Kayoko; Maeda Takeyasu
 CORPORATE SOURCE: Division of Oral Anatomy, Niigata University Graduate School of Medical and Dental Sciences, 2-5274 Gakkocho-dori, Niigata 951-8514, Japan.
 SOURCE: Neuroscience letters, (2007 Feb 2) Vol. 412, No. 3, pp. 222-6. Electronic Publication: 2006-12-18.
 Journal code: 7600130. ISSN: 0304-3940.
 PUB. COUNTRY: Ireland
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200704
 ENTRY DATE: Entered STN: 24 Jan 2007
 Last Updated on STN: 6 Apr 2007
 Entered Medline: 5 Apr 2007

AB Our recent study revealed an intense immunoreaction for GDNF and its

receptors in the Ruffini endings, primary mechanoreceptors in the periodontal ligament, of young rats. However, no information is available for the expression of GDNF and its receptors during their development. The present study aimed to reveal postnatal changes in the immuno-expression of GDNF, GFRalpha and RET in the periodontal Ruffini endings of the rat incisors by double immunofluorescent staining. At postnatal day 3 (PO 3d), no structure with GDNF-, GFRalpha-, or RET-immunoreaction existed in the periodontal ligament. The PGP 9.5-positive nerve fibers without GDNF- and RET-immunoreaction displayed a dendritic fashion at PO 1w, with a GFRalpha-reaction found around these nerves. At PO 2w, GDNF-positive terminal Schwann cells occurred near the thick and dendritic axons, a part of which showed a RET-reaction, with no reactive cells near the thin nerves. The terminal Schwann cells became positive for GFRalpha, but lacked RET-immunoreaction. At PO 3w, when the formation of the periodontal Ruffini endings had proceeded, GDNF-positive terminal Schwann cells began to increase in number. This stage-specific immuno-expression pattern suggests that GDNF is a key molecule for the maturation and maintenance of the periodontal Ruffini endings.

L4 ANSWER 11 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:635003 CAPLUS

DOCUMENT NUMBER: 145:90170

TITLE: Tissue engineering devices for the repair and regeneration of tissue

INVENTOR(S): Seyda, Agnieszka; Colter, David C.; Buensuceso, Charito S.; Sridevi, Dhanaraj; Gosiewska, Anna; Geesin, Jeffrey C.; Scopelianos, Angelo G.

PATENT ASSIGNEE(S): Ethicon, Inc., USA

SOURCE: PCT Int. Appl., 32 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006068972	A2	20060629	WO 2005-US45732	20051215
WO 2006068972	A3	20070503		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
US 20060171930	A1	20060803	US 2005-297156	20051208
AU 2005319401	A1	20060629	AU 2005-319401	20051215
CA 2591979	A1	20060629	CA 2005-2591979	20051215
EP 1835949	A2	20070926	EP 2005-854448	20051215
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
JP 2008523957	T	20080710	JP 2007-548345	20051215
PRIORITY APPLN. INFO.:			US 2004-637984P	P 20041221
			WO 2005-US45732	W 20051215

AB Tissue engineering devices for use in the repair or regeneration of tissue made of support scaffolds and cell sheets. The seeding of cells on scaffolds using cell sheets was compared against the conventional method of seeding scaffolds with cell suspension. Seeding of a biodegradable, biocompatible scaffold with cell sheets provides equivalent cell viability and cell distribution to seeding by cell suspension. Therefore, seeding scaffolds using the cell sheet method is a good alternative to the conventional method of cell suspension seeding with the benefit of one-step application and more controlled device handling.

L4 ANSWER 12 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:681122 CAPLUS
DOCUMENT NUMBER: 145:130940
TITLE: Tissue engineering devices with polymeric support scaffolds for the repair and regeneration of tissue
INVENTOR(S): Seyda, Agnieszka; Colter, David C.; Buensuceso, Charito S.; Dhanaraj, Sridevi; Gosiewska, Anna; Geesin, Jeffrey C.; Scopellianos, Angelo
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 12 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20060153815	A1	20060713	US 2005-304091	20051215
PRIORITY APPLN. INFO.:			US 2004-637781P	P 20041221

AB Tissue engineering devices for use in the repair or regeneration of tissue made of support scaffolds and cell sheets are described. Thus, cells were subjected to cell sheet preparation and were combined with nonwoven, degradable scaffolds as a first step towards generation of tissue engineering devices. Osteoblasts/chondrocytes for musculoskeletal applications as well as urothelial cells/bladder smooth muscle cells for urogenital applications were tested. Cells were cultured prior to scaffold deposition, e.g., in sheets on thermoresponsive poly(NIPAAm)-coated dishes.

L4 ANSWER 13 OF 41 MEDLINE on STN DUPLICATE 8

ACCESSION NUMBER: 2006301803 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16671879
TITLE: Profiling the cytokines in gingival crevicular fluid using a cytokine antibody array.
AUTHOR: Sakai Akihiko; Ohshima Mitsuhiro; Sugano Naoyuki; Otsuka Kichibee; Ito Koichi
CORPORATE SOURCE: Department of Periodontology, Nihon University School of Dentistry, Tokyo, Japan.
SOURCE: Journal of periodontology, (2006 May) Vol. 77, No. 5, pp. 856-64.
Journal code: 8000345. ISSN: 0022-3492.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Dental Journals; Priority Journals
ENTRY MONTH: 200607
ENTRY DATE: Entered STN: 31 May 2006
Last Updated on STN: 20 Jul 2006
Entered Medline: 19 Jul 2006

AB BACKGROUND: Various compounds have been detected in gingival crevicular

fluid (GCF) as indicators of periodontal disease activity. Therefore, the analysis of GCF may be especially beneficial for diagnosing current periodontal status and addressing the effects of treatment. Moreover, the identification of new markers in GCF may also contribute to elucidating novel mechanisms involved in periodontal disease. This study sought novel marker proteins specific to chronic periodontitis by profiling cytokines in GCF using a cytokine antibody array system. METHODS: Human cytokine array V, which detects 79 cytokines on one membrane, was used to determine the profile of cytokines in GCF from seven subjects with chronic periodontitis and seven subjects with healthy periodontia. The profile was exposed to x-ray film and quantified using image analysis software. Healthy and diseased sites were compared statistically. RESULTS: We detected 10 cytokines in periodontally healthy sites and 36 cytokines in periodontally diseased sites. Interleukin-8 (IL-8) and transforming growth factor-beta 2 (TGF-beta2) were detected at high levels in healthy and diseased subjects. There were significant differences between healthy and diseased subjects in the levels of tissue inhibitor of metalloproteinases-2 (TIMP-2), tumor necrosis factor-beta (TNF-beta), growth-related oncogene (GRO), interferon-inducible protein-10 (IP-10), angiogenin (Ang), vascular endothelial growth factor (VEGF), insulin-like growth factor binding protein-3 (IGFBP-3), osteoprotegerin (OPG), epidermal growth factor (EGF), glial-derived neurotrophic factor (GDNF), pulmonary and activation-regulated chemokine (PARC), oncostatin M (OSM), fibroblast growth factor-4 (FGF-4), IL-16, homologous to lymphotoxins (LIGHT), and placenta growth factor (PlGF). Of these, the newly detected cytokines were GRO, Ang, IGFBP-3, GDNF, PARC, OSM, FGF-4, IL-16, LIGHT, and PlGF. CONCLUSIONS: In this study, we detected several cytokines in GCF using a cytokine antibody array system, including both inflammatory cytokines and various growth factors. Therefore, periodontal disease may participate in the wound healing process and in tissue destruction via the inflammatory process. Our results suggest that the quantification of these cytokines in GCF provides useful information for the diagnosis of periodontal disease status.

L4 ANSWER 14 OF 41 MEDLINE on STN DUPLICATE 9
 ACCESSION NUMBER: 2006255023 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 16513266
 TITLE: Expression of GDNF and its receptors in the periodontal mechanoreceptor.
 AUTHOR: Aita Megumi; Kawano Yoshiro; Maeda Takeyasu
 CORPORATE SOURCE: Division of Oral Anatomy, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan.. aitam@dent.niigata-u.ac.jp
 SOURCE: Neuroscience letters, (2006 May 29) Vol. 400, No. 1-2, pp. 25-9. Electronic Publication: 2006-03-02. Journal code: 7600130. ISSN: 0304-3940.
 PUB. COUNTRY: Ireland
 DOCUMENT TYPE: (COMPARATIVE STUDY)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200608
 ENTRY DATE: Entered STN: 9 May 2006
 Last Updated on STN: 5 Aug 2006
 Entered Medline: 4 Aug 2006
 AB Our previous studies have revealed the involvement of signaling pathways of BDNF and NT-4/5 via TrkB in the development, regeneration, survival and maintenance of the Ruffini endings, primary mechanoreceptors in the periodontal ligament. However, the involvement of other neurotrophins remains unclear. The present study examined the expression of GDNF, GFRalpha3, and RET in the incisor periodontal

ligament and trigeminal ganglion of young rats by RT-PCR and immunocytochemistry. All these mRNAs were detected in both tissues by RT-PCR. These immunoreactions were found in the terminal Schwann cells associated with the periodontal Ruffini endings, as confirmed by histochemistry for non-specific cholinesterase activity. Their axonal branches showed GFRalphal- and RET-immunoreactions but lacked GDNF-immunoreactivity. In the trigeminal ganglion, about 30% of the neurons were immunoreactive to GFRalphal and RET. Averages of cross-sectional areas of their positive neurons demonstrated that they could mainly be categorized as medium-sized neurons. GDNF-immunoreaction was restricted to the satellite cells and not in trigeminal ganglion neurons. These findings indicate that GDNF mediates trophic effects on the survival and target innervation of the periodontal Ruffini endings via GFRalphal and RET.

L4 ANSWER 15 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2005:259902 CAPLUS

DOCUMENT NUMBER: 142:303690

TITLE: Remedy and therapeutic method for periodontal diseases and pulpal diseases with neurotrophic factors

INVENTOR(S): Kurihara, Hidemi; Kawaguchi, Hiroyuki; Takeda, Katsuhiko; Shiba, Hideki; Mizuno, Noriyoshi; Yoshino, Hiroshi; Hasegawa, Naohiko; Shinohara, Hiroaki

PATENT ASSIGNEE(S): Two Cells Co. Ltd., Japan

SOURCE: PCT Int. Appl., 86 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005025605	A1	20050324	WO 2004-JP13023	20040908
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
AU 2004271843	A1	20050324	AU 2004-271843	20040908
EP 1671641	A1	20060621	EP 2004-787706	20040908
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK			
CN 1871024	A	20061129	CN 2004-80031194	20040908
RU 2336089	C2	20081020	RU 2006-111465	20040908
US 20070071693	A1	20070329	US 2006-571069	20061207
PRIORITY APPLN. INFO.:			JP 2003-316719	A 20030909
			WO 2004-JP13023	W 20040908

AB It is intended to provide a remedy and a therapeutic method for periodontal diseases and pulpal diseases, a transplantation material for regenerating a periodontal tissue and a method of regenerating a periodontal tissue. Namely, a remedy for periodontal diseases and pulpal diseases comprising a neurotrophic factor as the active ingredient. The effect of brain-derived neurotrophic factor (BDNF) on cultured human

periodontal ligament cell and human gingival keratinocyte was
examined

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 16 OF 41 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 2005583578 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16259615
TITLE: Brain-derived neurotrophic factor enhances
periodontal tissue regeneration.
AUTHOR: Takeda Katsuhiko; Shiba Hideki; Mizuno Noriyoshi; Hasegawa
Naohiko; Mouri Yoshihiro; Hirachi Akio; Yoshino Hiroshi;
Kawaguchi Hiroyuki; Kurihara Hidemi
CORPORATE SOURCE: Department of Periodontal Medicine, Division of Frontier
Medical Science, Hiroshima University Graduate School of
Biomedical Sciences, Hiroshima, Japan.
SOURCE: Tissue engineering, (2005 Sep-Oct) Vol. 11, No. 9-10, pp.
1618-29.
Journal code: 9505538. ISSN: 1076-3279.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200512
ENTRY DATE: Entered STN: 3 Nov 2005
Last Updated on STN: 23 Dec 2005
Entered Medline: 22 Dec 2005

AB To address whether brain-derived neurotrophic factor (BDNF)
could be involved in periodontal tissue regeneration, we
examined the effects of BDNF on proliferation and the expression of bone
(cementum)- related proteins (osteopontin, bone morphogenetic protein
[BMP]-2, type I collagen, alkaline phosphatase [ALPase], and osteocalcin)
in cultures of human periodontal ligament (HPL) cells, which are
thought to be prerequisite for periodontal tissue regeneration,
and on proliferation and angiogenesis in human endothelial cells.
Furthermore, we examined the effect of BDNF on the regeneration of
periodontal tissues in experimentally induced periodontal
defects in dogs. BDNF elevated the expression of ALPase and osteocalcin
mRNAs and increased the synthesis of osteopontin, BMP-2, and type I
collagen DNA in HPL cells. BDNF stimulated mRNA expression of vascular
endothelial growth factor-B and tenascin-X, and proliferation and
angiogenesis in human endothelial cells. In vivo studies showed that BDNF
stimulated the formation of new alveolar bone cementum and connective new
fibers, which were inserted into the newly formed cementum and bone. BDNF
also stimulated blood capillary formation. These findings suggest that
the regulation of functioning of periodontal ligament cells and
endothelial cells by BDNF results in the promotion of periodontal
tissue regeneration.

L4 ANSWER 17 OF 41 MEDLINE on STN DUPLICATE 11
ACCESSION NUMBER: 2006089651 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16477147
TITLE: Neurotrophin-4/5-depletion induces a delay in
maturation of the periodontal Ruffini endings in
mice.
AUTHOR: Maruyama Yuko; Harada Fumiko; Jabbar Shahiqul; Saito Isao;
Aita Megumi; Kawano Yoshiro; Suzuki Akiko; Nozawa-Inoue
Kayoko; Maeda Takeyasu
CORPORATE SOURCE: Divisions of Oral Anatomy, Department of Oral Biological
Science, Niigata University Graduate School of Medical and

SOURCE: Dental Sciences, Niigata, Japan.
 Archives of histology and cytology, (2005 Dec) Vol. 68, No. 4, pp. 267-88.
 Journal code: 8806082. ISSN: 0914-9465.
 PUB. COUNTRY: Japan
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200901
 ENTRY DATE: Entered STN: 15 Feb 2006
 Last Updated on STN: 12 Dec 2006
 Entered Medline: 29 Jan 2009

AB Neurotrophin-4/5 (NT-4/5) - a member of the neurotrophic factors - is a ligand for TrkB, which has been reported to be expressed in the mechanoreceptive Ruffini endings of the periodontal ligament. The present study examined developmental changes in the terminal morphology and neural density in homozygous mice with a targeted disruption of the nt-4/5 gene and wild-type mice by immunohistochemistry for protein gene product 9.5 (PGP 9.5), a general neuronal marker, and by quantitative analysis using an image analyzer. Postnatal development of terminal Schwann cells was also investigated by enzymatic histochemistry for non-specific cholinesterase activity (ChE). Furthermore, the immuno-expression of TrkB and low affinity nerve growth factor receptor (p75-NGFR) was surveyed in the periodontal Ruffini endings as well as trigeminal ganglion. At postnatal 1 week, the lingual periodontal ligament of both types of mice contained PGP 9.5-positive nerve fibers showing a tree-like ramification with axonal swellings in their course. In both types of mice at 2 weeks of age, comparatively thick nerve fibers with a smooth outline increased in number, and frequently ramified to form nerve terminals with dendritic profiles. However, no typical Ruffini endings with irregular outlines observed in the adult wild-type mice were found in the periodontal ligament at this stage. At postnatal 3 weeks, typical Ruffini endings with irregular outlines were discernable in the periodontal ligament of the wild-type mice while the dendritic endings showing smooth outlines were restricted to the homozygous mice. After postnatal 8 weeks, both types of mice showed an increase in the number of Ruffini endings, but the morphology differed between the wild-type and NT-4/5 homozygous mice. In the wild-type mice, a major population of the Ruffini endings expanded their axonal branches and developed many microprojections, resulting in a reduction of endings with smooth outlines. In contrast, we failed to find such typical Ruffini endings in the periodontal ligament of the homozygous mice: A majority of the periodontal Ruffini endings continued to show smooth outlines at postnatal 12 weeks. Quantitative analysis on neural density demonstrated a reduction in the homozygous mice with a significant difference by postnatal 8 weeks. Enzymatic histochemistry for non-specific ChE did not exhibit a distinct difference in the distribution and density of terminal Schwann cells between wild-type and homozygous mice. Furthermore, TrkB and p75-NGFR mRNA and proteins did not differ in the trigeminal ganglion between the two types. The periodontal Ruffini endings also displayed immunoreactivities for TrkB and p75-NGFR in both phenotypes. These findings suggest that the nt-4/5 gene depletion caused a delay in the formation and maturation of the periodontal Ruffini endings in the mice by inhibiting the growth of the periodontal nerves at an early stage, and indicate that multiple neurotrophins such as NT-4/5 and BDNF might play roles in the development and/or maturation of the periodontal Ruffini endings.

DOCUMENT NUMBER: 145:394244
TITLE: Expression of NGF and trkA mRNAs in dogs' periodontal tissue with traumatic occlusion
AUTHOR(S): Dong, Yan; Liu, Hongchen; Wang, Xinmu; Wu, Shengxi
CORPORATE SOURCE: Department of Stomatology, General Hospital of PLA, Beijing, 100853, Peop. Rep. China
SOURCE: Kouqiang Yixue (2005), 25(4), 216-218
CODEN: KYOIAY; ISSN: 1003-9872
PUBLISHER: Kouqiang Yixue Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The occlusal surface of the right first and second maxillary molars in 18 dogs were unilaterally raised 1.5 mm with the casting Ni-Cr inlay which were fixed in Class I inlay hole. On the 3rd, 7th, 14th, 30th and 60th days after teeth operations, the upper and lower molars in two-side dentition were extracted. Periodontium tissue was detached from root cementum. Nerve growth factor (NGF) and tyrosine kinase A (trkA) mRNAs were detected by using RT-PCR in exptl. and control groups. NGF mRNA expression up-regulated from the 3rd to 30th days compared with the control group and reached peak level during 14 to 30 days after traumatic occlusion was induced. Compared with contralateral side and control group, NGF mRNA was about three-fold on day 30 in trauma periodontium ligament. An upregulation expression of NGF mRNA in contralateral sides was also observed during 3 to 7 days. TrkA mRNA expression was similar to that of NGF mRNA and had the highest level on the 30th day after teeth operation. NGF and trkA mRNAs at the trauma periodontium side were stronger than that at the contralateral side and control group. The present study showed that NGF and trkA mRNAs were increased in traumatic occlusal periodontal tissue. A unilateral occlusion initiated nerve responses in the whole molar dentition. NGF might play an important role in orofacial pain caused by traumatic occlusion.

L4 ANSWER 19 OF 41 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:116637 BIOSIS
DOCUMENT NUMBER: PREV200500115225
TITLE: The involvement of BDNF in development/regeneration of the periodontal Ruffini ending.
AUTHOR(S): Maeda, T. [Reprint Author]
CORPORATE SOURCE: Div Oral AnatGrad Sch Med Dent Sci, Niigata Univ, Niigata, Japan
maedat@dent.niigata-u.ac.jp
SOURCE: Anatomical Science International, (August 2004) Vol. 79, No. August, pp. 78. print.
Meeting Info.: 16th International Congress of the IFAA (International Federation of Associations of Anatomists) and the 109th Annual Meeting of the Japanese Association of Anatomists. Kyoto, Japan. August 22-27, 2004. Japanese Association of Anatomists; International Federation of Associations of Anatomists.
ISSN: 1447-6959 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Mar 2005
Last Updated on STN: 23 Mar 2005

L4 ANSWER 20 OF 41 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:463163 BIOSIS
DOCUMENT NUMBER: PREV200510243637
TITLE: Endoneural fibroblasts isolation and culture.

Original Title: Aislamiento y cultivo de fibroblastos endoneurales.
 AUTHOR(S): Leau, Leslie [Reprint Author]; Perdomo, Sandra; Spinel, Clara
 CORPORATE SOURCE: Univ Nacl Colombia, Fac Ciencias, Dept Biol, Bogota, Colombia
 SOURCE: Acta Biologica Colombiana, (2004) Vol. 9, No. 2, pp. 57-65. ISSN: 0120-548X.
 DOCUMENT TYPE: Article
 LANGUAGE: Spanish
 ENTRY DATE: Entered STN: 9 Nov 2005
 Last Updated on STN: 9 Nov 2005

AB Fibroblasts which are tissue-specific, constantly degrade and synthesize the different elements of the extra-cellular matrix (ECM), while at the same time remodel tissues that are being repaired. Dermal fibroblasts are well studied both in vitro and in vivo, and are used to regenerate dermal EMC which in turn supports the regeneration of the epidermis. Confluence of dermal or periodontal fibroblasts takes place between 8 and 10 days of culture. In the process of regeneration of damaged peripheral nerves, Schwann's cells secrete neurotrophic and neurotropic growth factors and some of the EMC elements needed for regeneration to take place, which makes them the most studied and used cells in culture. So far, endoneural fibroblasts (EF) have not been considered as important elements in nerve regeneration, mainly because they may occasionally form fibromes that hinder regeneration. But there is evidence that they may play a role in the remodeling of the EMC, through the secretion of metalloproteins that modify the pre-Nerve Growth Factor (preNGF) secreted by Schwann's cells into active NGF, which promotes neurites regeneration. The aim of this study was to isolate EF from sciatic nerves taken from mature rats, and to obtain them in purified culture. A number of methods of dissection and digestion were developed to obtain primary pure EF cultures as well as to study them in the way Schwann's cells have been studied. Selective isolation of EF was accomplished, reaching confluence between the fourth and the fifth day in monolayer primary culture. Producing a population of EF will make it possible to carry out studies in tridimensional culture and in prosthesis in order to define and develop new alternatives for the regeneration of peripheral nerves.

L4 ANSWER 21 OF 41 MEDLINE on STN DUPLICATE 12
 ACCESSION NUMBER: 2003385376 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 12923891
 TITLE: Involvement of brain-derived neurotrophic factor (BDNF) in the development of periodontal Ruffini endings.
 AUTHOR: Hoshino Natalia; Harada Fumiko; Alkhamrah Bashar Anas; Aita Megumi; Kawano Yoshio; Hanada Kooji; Maeda Takeyasu
 CORPORATE SOURCE: Department of Oral Biological Science, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan.
 SOURCE: The anatomical record. Part A, Discoveries in molecular, cellular, and evolutionary biology, (2003 Sep) Vol. 274, No. 1, pp. 807-16.
 Journal code: 101234285. ISSN: 1552-4884.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200405
 ENTRY DATE: Entered STN: 19 Aug 2003
 Last Updated on STN: 22 May 2004
 Entered Medline: 21 May 2004

AB The periodontal Ruffini ending has been reported to show immunoreactivity for tyrosine kinase B (trkB), the high-affinity receptor for brain-derived neurotrophic factor (BDNF), in the periodontal ligament of the rat incisor. Furthermore, adult heterozygous BDNF-mutant mice showed malformation and reduction of the periodontal Ruffini endings. To investigate further roles of BDNF in these structures, the development, distribution, and terminal morphology of Ruffini endings were examined in the incisor periodontal ligament of heterozygous and homozygous BDNF mutant mice, as well as in the wild-type littermate by immunohistochemistry for protein gene product (PGP) 9.5, a general neuronal marker. A similar distribution and terminal formation of PGP 9.5-immunoreactive nerve fibers was recognized in the periodontal ligament of all phenotypes at postnatal week (PW) 1. At this stage, the nerve fibers had a beaded appearance, but did not form the periodontal Ruffini endings. At PW2, the heterozygous and wild-type mice started to show ramified nerve fibers resembling the mature shape of periodontal Ruffini endings. At PW3, the Ruffini endings occurred in the periodontal ligament of the wild-type and heterozygous mice. While the Ruffini endings of the wild-type mice appeared either ruffled or smooth, as reported previously, most of these structures showed a smooth outline in the heterozygous mice. The homozygous mice lacked the typical Ruffini endings at PW3. In the quantitative analysis, homozygous mice had the smallest percentages of PGP 9.5-immunoreactive areas at the same postnatal periods, but there were no significant differences between wild-type and heterozygous mice during PW1-3. These findings suggest a possible involvement of BDNF during the postnatal development and, in particular, the maturation of periodontal Ruffini endings. Furthermore, other neurotrophins may play a role in the development and/or early maturation of the periodontal nerve fibers, as indicated by the presence of nerve fibers in the BDNF-homozygous mice.

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L4 ANSWER 22 OF 41 MEDLINE on STN DUPLICATE 13
 ACCESSION NUMBER: 2003316788 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 12846558
 TITLE: The involvement of brain-derived neurotrophic factor (BDNF) in the regeneration of periodontal Ruffini endings following transection of the inferior alveolar nerve.
 AUTHOR: Harada Fumiko; Hoshino Natalia; Hanada Kooji; Kawano Yoshiro; Atsumi Yukako; Wakisaka Satoshi; Maeda Takeyasu
 CORPORATE SOURCE: Division of Oral Anatomy, Department of Oral Biological Science, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan.
 SOURCE: Archives of histology and cytology, (2003 May) Vol. 66, No. 2, pp. 183-94.
 Journal code: 8806082. ISSN: 0914-9465.
 PUB. COUNTRY: Japan
 DOCUMENT TYPE: (COMPARATIVE STUDY)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200310
 ENTRY DATE: Entered STN: 9 Jul 2003
 Last Updated on STN: 8 Oct 2003
 Entered Medline: 7 Oct 2003

AB The present study employed immunohistochemistry for protein gene product 9.5 (PGP 9.5) to examine the regeneration process of Ruffini endings, the primary mechanoreceptor in the periodontal ligament, in heterozygous mice with targeted disruption of the brain-derived

neurotrophic factor (BDNF) gene and their littermates, following transection of the inferior alveolar nerve. When immunostained for PGP 9.5, periodontal Ruffini endings appeared densely distributed in the periodontal ligament of the heterozygous mice, but the density of the positively stained nerve fibers in the ligament was 20% lower than that in the control littermates. At 3 days after surgery, the PGP 9.5-positive neural elements had disappeared; they began to appear in the periodontal ligament of both animals at 7 days. However, the recovery pattern of the PGP 9.5-positive nerves differed between heterozygous and wild type mice, typical periodontal Ruffini endings morphologically identical to those in the control group appeared in the wild-type mice at 7 days, whereas such Ruffini endings were detectable in the heterozygous mice at 28 days, though much smaller in number. On day 28, when PGP 9.5-positive nerves were largely regenerated in wild type mice, their distribution was much less dense in the ligament of the heterozygous mice than in the non-treated heterozygous mice. The density of PGP 9.5-positive nerve fibers was significantly lower in the heterozygous mice than in wild type mice at any stage examined. These data showing that a reduced expression of BDNF causes delayed regeneration of the periodontal Ruffini endings suggest the involvement of BDNF in the regeneration process of these mechanoreceptors.

L4 ANSWER 23 OF 41 MEDLINE on STN DUPLICATE 14
 ACCESSION NUMBER: 2003081727 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 12593600
 TITLE: Neurotrophins in cultured cells from periodontal tissues.
 AUTHOR: Kurihara Hidemi; Shinohara Hiroaki; Yoshino Hiroshi; Takeda Katsuhiko; Shiba Hideki
 CORPORATE SOURCE: Department of Periodontal Medicine, Division of Frontier Medical Science, Hiroshima University Graduate School of Biomedical Science, Hiroshima, Japan..
 SOURCE: Journal of periodontology, (2003 Jan) Vol. 74, No. 1, pp. 76-84. Ref: 67
 Journal code: 8000345. ISSN: 0022-3492.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 General Review; (REVIEW)
 LANGUAGE: English
 FILE SEGMENT: Dental Journals; Priority Journals
 ENTRY MONTH: 200305
 ENTRY DATE: Entered STN: 21 Feb 2003
 Last Updated on STN: 8 May 2003
 Entered Medline: 7 May 2003
 AB We review the basic functions of neurotrophins and their receptors and discuss the expression and functions of neurotrophins and their specific receptors based on recent data using cultured cells from human periodontal tissues. Neurotrophins, nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), and neurotrophin-3 (NT-3) play crucial roles in the differentiation and survival of neural cells. Neurotrophins activate 2 different receptor classes: the tropomyosin-related kinase (Trk) family of receptor tyrosine kinases (TrkA, TrkB, and TrkC) and the p75 receptor, a member of the tumor necrosis factor receptor superfamily. Neurotrophins regulate both cell death and cell survival through activations of Trk receptors and/or p75 neurotrophin receptor. It has been reported that neurotrophins are also produced from non-neuronal cells, such as leukocytes, osteoblasts, or fibroblasts, and act in many other ways on non-neuronal cells. Neurotrophin expression during bone

fracture healing is especially interesting, and neurotrophins are now implicated in hard tissue regeneration. It is well known that neurotrophins and their receptors are expressed in tooth development. Recent studies have found that neurotrophins and Trk receptors are expressed in mouse osteoblastic cell lines. Human periodontal ligament cells, human gingival fibroblasts, and human gingival keratinocytes expressed mRNA for NGF and TrkA. The secretion of bioactive NGF peptides from human periodontal ligament cells and human gingival keratinocytes was confirmed by bioassay using PC12 cells (rat adrenal pheochromocytoma cells). The expression of NGF and TrkA.mRNA was regulated by interleukin (IL)-1beta. NGF increased DNA synthesis and expressions of mRNA for bone-related proteins, alkaline phosphatase, and osteopontin in human periodontal ligament cells. Neurotrophins and Trk receptors expressed in human periodontal tissue may contribute to regeneration as well as innervation of periodontal tissue through local autocrine and paracrine pathways. Recent data suggest that some functions of neurotrophins and Trk receptors relate to periodontal disease and periodontal tissue regeneration. However, in vivo studies will be required to clarify the roles of neurotrophins and their receptors, including p75, in periodontal disease and periodontal tissue regeneration.

L4 ANSWER 24 OF 41 MEDLINE on STN DUPLICATE 15
 ACCESSION NUMBER: 2003184360 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 12703556
 TITLE: The periodontal Ruffini endings in brain derived neurotrophic factor (BDNF) deficient mice.
 AUTHOR: Alkhamrah Bashar Anas; Hoshino Natalia; Kawano Yoshiro; Harada Fumiko; Hanada Kooji; Maeda Takeyasu
 CORPORATE SOURCE: Divisions of Oral Anatomy, Department of Oral Biological Science, Niigata University Graduate School of Medical and Dental Sciences, Gakkocho-dori, Niigata, Japan.
 SOURCE: Archives of histology and cytology, (2003 Mar) Vol. 66, No. 1, pp. 73-81.
 Journal code: 8806082. ISSN: 0914-9465.
 PUB. COUNTRY: Japan
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200311
 ENTRY DATE: Entered STN: 22 Apr 2003
 Last Updated on STN: 17 Dec 2003
 Entered Medline: 19 Nov 2003

AB Innervation and terminal morphology in the lingual periodontal ligament of the incisor were investigated in brain derived neurotrophic factor (BDNF) heterozygous mice and littermate wild-type mice (aged two months) using immunohistochemistry for protein gene product 9.5 (PGP 9.5), a general neuronal marker. In addition, computer-assisted quantitative analysis was performed for a comparison of neuronal density in the periodontal ligament between heterozygous and wild-type mice. In wild-type mice, the periodontal ligament was found to be richly innervated by the mechanoreceptive Ruffini endings and nociceptive free nerve endings in the alveolus-related part of the periodontal ligament. The periodontal Ruffini endings in the wild-type mice incisor ligament were classified into two types: type I with ruffled outlines, and type II with a smooth outline. BDNF heterozygous mice showed malformations of the type I Ruffini endings which included fewer nerve fibers and fewer ramifications than those in wild-type mice as well as smooth outlines of the axon terminals. Quantitative analysis under a confocal microscope

showed a roughly 18% reduction in neuronal density in the periodontal ligament of the heterozygous mice. These findings suggest that the development and maturation of the periodontal Ruffini endings require BDNF.

L4 ANSWER 25 OF 41 MEDLINE on STN DUPLICATE 16
ACCESSION NUMBER: 2002646060 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12396578
TITLE: Ruffini endings are absent from the periodontal ligament of trkB knockout mice.
AUTHOR: Matsuo Saburou; Ichikawa Hiroyuki; Silos-Santiago Inmaculada; Kiyomiya Ken-ichi; Kurebe Masaru; Arends Joop J A; Jacquin Mark F
CORPORATE SOURCE: Department of Toxicology, Veterinary Science, Osaka Prefecture University Graduate School of Agriculture and Biological Sciences, Sakai, Japan.
SOURCE: Somatosensory & motor research, (2002) Vol. 19, No. 3, pp. 213-7.
Journal code: 8904127. ISSN: 0899-0220.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 31 Oct 2002
Last Updated on STN: 18 Mar 2003
Entered Medline: 17 Mar 2003
AB To clarify the role of neurotrophin receptors in the development of Ruffini endings, periodontal ligaments and trigeminal ganglia of trkA, trkB, and trkC knockout mice were immunostained for protein gene product 9.5 (PGP 9.5), calcitonin gene-related peptide (CGRP), parvalbumin (PV), and calretinin (CR). Innervation patterns of PGP 9.5- and CGRP-immunoreactive fibers were examined in the periodontal ligament of the knockout mice. PGP 9.5-positive fibers in the incisal periodontal ligaments of trkA and trkC knockout mice form Ruffini endings distinguished by dendritic ramifications and branches. However, Ruffini endings were not present in the periodontal ligament of trkB knockout mice. Only free nerve endings were observed in tissue of trkB knockout mice. Compared with trkA and trkC knockouts, the proportion of CR-positive neurons in mandibular and maxillary regions of the trigeminal ganglion of trkB knockout mice is decreased. These findings indicate that the development of periodontal Ruffini endings is regulated by trkB-dependent and CR-coexpressing neurons.

L4 ANSWER 26 OF 41 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:380428 BIOSIS
DOCUMENT NUMBER: PREV200300380428
TITLE: INVOLVEMENT OF BDNF IN THE DEVELOPMENT OF THE PERIODONTAL RUFFINI ENDINGS IN THE MOUSE INCISOR.
AUTHOR(S): Hoshino, N. [Reprint Author]; Harada, F. [Reprint Author]; Kawano, Y. [Reprint Author]; Hanada, K. [Reprint Author]; Yamamura, K. [Reprint Author]; Maeda, T. [Reprint Author]
CORPORATE SOURCE: Oral Anatomy, Orthodontics, Physiology, Niigata University, Niigata, Japan
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 849.3.
<http://sfn.scholarone.com.cd-rom>.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Aug 2003
Last Updated on STN: 20 Aug 2003

AB Previous studies had shown TrkB immunoreactivity in the periodontal Ruffini endings (PRE) of the rat incisor ligament, leading us to speculate that the development and/or maintenance of these mechanoreceptors require BDNF. In the present study, the distribution and morphology of the PRE were investigated in the incisor ligament of the BDNF mutant mice by immunocytochemistry for protein gene product 9.5 (PGP 9.5; a general neuronal marker). Wild and homozygous BDNF-KO mice were anesthetized and perfused with 4% paraformaldehyde in 0.1M phosphate buffer. Maxillae, including incisors, were removed, decalcified, and frozen sections were sagittally cut at a thickness of 35mm. Then, they were processed using the ABC method. The PRE, displaying a dendritic fashion, were observed in the alveolar half of the incisor ligament in both typed mice. In the (-/-) mice, however, the PRE showed significant less extensive arborizations than in the (+/+) mice. Besides, the density of the PRE appeared lower than in the wild type littermates. These data indicate that the depletion of BDNF affected the terminal arborization and innervation density of the PRE implying an important role for BDNF in the development and maintenance of these structures. Furthermore, since not all PRE disappeared in the homozygous mice, other neurotrophins, such as NT4/5, might as well be involved in their development and survival.

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ACCESSION NUMBER: 2003:380429 BIOSIS
DOCUMENT NUMBER: PREV200300380429
TITLE: DEPLETION OF BDNF INDUCES DELAY OF REGENERATION OF THE PERIODONTAL RUFFINI ENDINGS.
AUTHOR(S): Harada, F. [Reprint Author]; Maeda, T. [Reprint Author]; Hoshino, N. [Reprint Author]; Iijima, K. [Reprint Author]; Kawano, Y. [Reprint Author]; Hanada, K.; Atsumi, Y.; Wakisaka, S.
CORPORATE SOURCE: Oral Anatomy, Orthodontics, Niigata University, Niigata, Japan
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 849.4.
<http://sfn.scholarone.com.cd-rom>.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Aug 2003
Last Updated on STN: 20 Aug 2003

AB The periodontal Ruffini endings have been reported to show immunoreactivity for TrkB, a receptor for brain derived neurotrophic factor (BDNF), suggesting its involvement in development/regeneration of these receptors. In this study, we investigated the regeneration process of the periodontal Ruffini endings (PRE) in heterozygous mice with target disruption of BDNF gene. Transection of the inferior alveolar nerve (IAN) was performed in the heterozygous and littermate wild-type mice. The cut ends of IAN were returned into the mandibular canal, and the wound was sutured. The animals were allowed to survive for 3, 7, 10, 14, 21 and 28 days. After each determined period, they were perfused transcardially with 4% paraformaldehyde in 0.1 M phosphate buffer. After decalcification of

mandibles including incisors, serial frozen sections were cut at a thickness of 30 μ m. Neural elements in the lingual ligament were demonstrated by immunohistochemistry for PGP 9.5, a general neuronal marker. In the wild-type mice, the regeneration of the PRE completed around postoperative 21 days, consistent with our previous reports. In the heterozygous mice, on the other hand, the regeneration of the PRE delayed. The lower density and malformation of the regenerated PRE were recognized even at postoperative 28 days. These findings indicated that the depletion of BDNF induced delay of the regeneration of the PRE, suggesting that they require BDNF for regeneration.

L4 ANSWER 28 OF 41 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:548165 BIOSIS
DOCUMENT NUMBER: PREV200100548165
TITLE: The Periodontal Ruffini Endings in the BDNF knock-out mouse.
AUTHOR(S): Hoshino, N. [Reprint author]; Alkhamrah, B. [Reprint author]; Hanada, K. [Reprint author]; Maeda, T. Orthodontics, Niigata University, Niigata, Japan
CORPORATE SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 1619. print.
SOURCE: Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Nov 2001
Last Updated on STN: 25 Feb 2002

AB We have previously shown TrkB immunoreactivity in the Periodontal Ruffini Endings (PRE) of the rat incisor ligament, leading us to speculate that the development and/or maintenance of these mechanoreceptors require BDNF. In the present study, the distribution and morphology of the PRE were investigated in the incisor ligament of the BDNF mutant mice by immunocytochemistry for protein gene product 9.5 (PGP 9.5; a general neuronal marker). Since homozygous BDNF-KO mice could only survive up to 2 weeks, we used heterozygous (+/-) and wild type (+/+) mice for this study. Animals were anesthetized and perfused with 4% paraformaldehyde in 0.1M phosphate buffer. Maxillae, including incisors, were removed, decalcified, and frozen sections were sagittally cut at a thickness of 40 μ m. Then, they were processed using the ABC method. The PRE, displaying a dendritic fashion, were observed in the alveolar half of the incisor ligament in both typed mice. In the (+/-) mice, however, the PRE showed less extensive arborizations than in the (+/+) mice. Furthermore, the density of the PRE appeared lower than in the wild type littermates. These data indicate that the reduction of BDNF affected the terminal arborization and innervation density of the PRE implying an important role for BDNF in the development and maintenance of the PRE. Furthermore, since not all PRE disappeared in the heterozygous mice, other neurotrophins, such as NT4/5, might as well be involved in their development and survival.

L4 ANSWER 29 OF 41 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:539364 BIOSIS
DOCUMENT NUMBER: PREV200100539364
TITLE: The Krox-20 null mutation impacts the development of mesencephalic trigeminal neurons.
AUTHOR(S): Turman, J. E. [Reprint author]; De, S.; Nguyen, A. Q.; Shuler, C. F.

CORPORATE SOURCE: Dept of Biokinesiol, USC, Los Angeles, CA, USA
 SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 1619. print.
 Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
 ISSN: 0190-5295.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 21 Nov 2001
 Last Updated on STN: 25 Feb 2002

AB The Krox-20 null mutant is an interesting model to study the development of oral-motor circuits. In these mutants, rhombomeres 3 and 5 are not maintained during development leading to disruptions of oral-motor behaviors and subsequent perinatal death due to poor nutrient intake. We are interested in studying the role of Krox-20 gene expression in the development of neuromuscular circuitry underlying jaw movements. This study focuses on the impact of the Krox-20 mutation on mesencephalic trigeminal neurons as they have an important role in neural circuits responsible for jaw reflexes and rhythmical jaw movements. These cells innervate either jaw closer muscle spindles or mechanoreceptors of the periodontal ligament. We hypothesized that mesencephalic trigeminal neurons would be spared in Krox-20 null mutants because Krox-20 is expressed in rhombomeres 3 and 5 whereas mesencephalic trigeminal neurons are derived from the mesencephalic neural crest. Counterstaining with associated cell counting was used to investigate the impact of the Krox-20 mutation on mesencephalic trigeminal neuron development. Results show that the number of mesencephalic trigeminal neurons is significantly reduced at birth in Krox-20 null mutants. These results were unexpected due to the incongruency between Krox-20 gene expression and the origin of these cells. The reduction in mesencephalic trigeminal neurons maybe due to insufficient neurotrophic support or a consequence of the loss of rhombomere 3. In conclusion, the Krox-20 mutation impacts a subset of primary sensory neurons critical for the execution of oral-motor behaviors.

L4 ANSWER 30 OF 41 MEDLINE on STN DUPLICATE 17
 ACCESSION NUMBER: 2001296132 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 11379889
 TITLE: Mitogenic effects of neutrophins on a periodontal ligament cell line.
 AUTHOR: Tsuboi Y; Nakanishi T; Takano-Yamamoto T; Miyamoto M; Yamashiro T; Takigawa M
 CORPORATE SOURCE: Department of Orthodontics, Okayama University Dental School, Japan.
 SOURCE: Journal of dental research, (2001 Mar) Vol. 80, No. 3, pp. 881-6.
 Journal code: 0354343. ISSN: 0022-0345.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Dental Journals; Priority Journals
 ENTRY MONTH: 200106
 ENTRY DATE: Entered STN: 25 Jun 2001
 Last Updated on STN: 25 Jun 2001
 Entered Medline: 21 Jun 2001

AB Nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), and neurotrophin-3 (NT-3) are three representative neurotrophins responsible for the differentiation and survival of neurons, and their high-affinity receptors are tropomyosin-receptor-kinase

(TRK)A, TRKB, and TRKC, respectively. In this study, we investigated the expression of neurotrophins in a mouse periodontal ligament cell line (MPL), by reverse transcription-polymerase chain-reaction (RT-PCR) and enzyme-linked immunoabsorbent assay (ELISA). We also studied the expression of TRK receptors on MPL by immunostaining and the effects of neurotrophins on the proliferation of MPL, with a hypothesis of autocrine mechanism of neurotrophins. Each neurotrophin and TRK receptor was expressed, and neurotrophins enhanced the proliferation of MPL. These findings suggest that the MPL has functional neurotrophin receptors involved in an autocrine function of neurotrophins. The expression level of neurotrophins and TRKs showed the reverse pattern, and we propose an auto-regulatory mechanism of ligands and receptors in accordance with the level of synthesized neurotrophins.

L4 ANSWER 31 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2001:609225 CAPLUS

DOCUMENT NUMBER: 135:355792

TITLE: trkA modulation of developing somatosensory neurons in oro-facial tissues: tooth pulp fibers are absent in trkA knockout mice

AUTHOR(S): Matsuo, S.; Ichikawa, H.; Henderson, T. A.; Silos-Santiago, I.; Barbacid, M.; Arends, J. J. A.; Jacquin, M. F.

CORPORATE SOURCE: Neurology, Washington University School of Medicine, St. Louis, MO, 63110, USA

SOURCE: Neuroscience (Oxford, United Kingdom) (2001), 105(3), 747-760

CODEN: NRSCDN; ISSN: 0306-4522

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To investigate the nerve growth factor requirement of developing oro-facial somatosensory afferents, we have studied the survival of sensory fibers subserving nociception, mechanoreception or proprioception in receptor tyrosine kinase (trkA) knockout mice using immunohistochem. trkA receptor null mutant mice lack nerve fibers in tooth pulp, including sympathetic fibers, and showed only sparse innervation of the periodontal ligament. Ruffini endings were formed definitively in the periodontal ligament of the trkA knockout mice, although calcitonin gene-related peptide- and substance P-immunoreactive fibers were reduced in number or had disappeared completely. trkA gene deletion had also no obvious effect on the formation of Meissner corpuscles in the palate. In the vibrissal follicle, however, some mechanoreceptive afferents were sensitive for trkA gene deletion, confirming a previous report [Fundin et al. (1997) Dev. Biol. 190, 94-116]. Moreover, calretinin-pos. fibers innervating longitudinal lanceolate endings were completely lost in trkA knockout mice, as were the calretinin-containing parent cells in the trigeminal ganglion. These results indicate that trkA is indispensable for developing nociceptive neurons innervating oral tissues, but not for developing mechanoreceptive neurons innervating oral tissues (Ruffini endings and Meissner corpuscles), and that calretinin-containing, trkA dependent neurons in the trigeminal ganglion normally participate in mechanoreception through longitudinal lanceolate endings of the vibrissal follicle.

REFERENCE COUNT: 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 32 OF 41 MEDLINE on STN

ACCESSION NUMBER: 2001193959 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11163324

DUPLICATE 18

TITLE: Morphological variation in the tyrosine receptor kinase A-immunoreactive periodontal ligament epithelium of developing and mature rats.

AUTHOR: Woodnutt D A; Byers M R

CORPORATE SOURCE: Dental School, University of Washington, Seattle, WA 98195, USA.

CONTRACT NUMBER: DE05159 (United States NIDCR NIH HHS)
T35DE07150 (United States NIDCR NIH HHS)

SOURCE: Archives of oral biology, (2001 Feb) Vol. 46, No. 2, pp. 163-71.
Journal code: 0116711. ISSN: 0003-9969.

PUB. COUNTRY: England; United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LANGUAGE: English

FILE SEGMENT: Dental Journals; Priority Journals

ENTRY MONTH: 200104

ENTRY DATE: Entered STN: 10 Apr 2001
Last Updated on STN: 10 Apr 2001
Entered Medline: 5 Apr 2001

AB Tyrosine receptor kinase A (trkA) is the high-affinity receptor for nerve growth factor. It has been found in several non-neuronal cell types, indicating biological roles independent of neural function, as well as in the nervous system. An initial study demonstrated that an antibody to the full extracellular domain did not label periodontal ligament epithelium (PLE; also known as epithelial rests of Malassez), but that another antibody which recognises a truncated 41-kDa form of trkA did label PLE. Thus, truncated trkA-immunoreactive (-IR) PLE was further investigated here in developing molars of young rats, and in its mature form in adult rat molars, for its reaction to moderate or deep molar injuries, and for its appearance along the continuously erupting incisors of mature rats. In some of the adult rat molars we also analysed the association of nerve fibres with PLE using antibodies for p75 neurotrophin receptor or peripherin. Rat jaws were fixed with 4% formaldehyde and demineralised, and bound antibody was detected with avidin-biotin-peroxidase and diaminobenzidine or fluorescence procedures. Light microscopy showed great variation in the appearance of trkA-IR PLE and considerable morphological changes during the eruption of molars and incisors. By electron microscopy it was shown that trkA-IR was not uniformly distributed in PLE cells but rather was concentrated in the peripheral zones of each cell cluster. Tooth injury did not influence the form or occurrence of PLE unless there was specific destruction of a ligament region. Qualitative analyses of nerve fibres showed that they only rarely innervated PLE in adult rats, indicating that the truncated receptor has non-neuronal functions in this epithelium. These results suggest that neurotrophin growth factors, acting via truncated trkA receptors, affect the interactions between PLE cells and the periodontal ligament, with fewer PLE interactions with nerves. Furthermore, the expression of these receptors on PLE supports the possibility that these cells are active during tooth development and eruption rather than being merely passive remnants of the degenerating tooth sheath. The similar trkA-IR of PLE and junctional epithelium, as well as their structural association, suggests interactions between these two epithelia.

L4 ANSWER 33 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:241521 CAPLUS

DOCUMENT NUMBER: 132:289231

TITLE: Fusion proteins of functional domains of the transforming growth factor β family of proteins and their preparation, biol. activity and uses

INVENTOR(S): Oppermann, Hermann; Tai, Mei-Sheng; McCartney, John

PATENT ASSIGNEE(S): Stryker Corporation, USA
 SOURCE: PCT Int. Appl., 162 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 3
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000020607	A2	20000413	WO 1999-US23371	19991007
WO 2000020607	A3	20000706		
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6677432	B1	20040113	US 1999-374958	19990816
CA 2344974	A1	20000413	CA 1999-2344974	19991007
CA 2657302	A1	20000413	CA 1999-2657302	19991007
AU 2000011038	A	20000426	AU 2000-11038	19991007
AU 772930	B2	20040513		
EP 1117804	A2	20010725	EP 1999-954771	19991007
EP 1117804	B1	20070613		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2002526115	T	20020820	JP 2000-574702	19991007
JP 3971107	B2	20070905		
AT 364704	T	20070715	AT 1999-954771	19991007
JP 2005287515	A	20051020	JP 2005-136701	20050509
JP 2005320339	A	20051117	JP 2005-156477	20050527
JP 2005290016	A	20051020	JP 2005-178654	20050617
JP 2008237223	A	20081009	JP 2008-132454	20080520
JP 2008231125	A	20081002	JP 2008-162608	20080620
PRIORITY APPLN. INFO.:				
			US 1998-103418P	P 19981007
			US 1999-374958	A 19990816
			US 1999-374936	A 19990816
			US 1999-375333	A 19990816
			CA 1999-2345024	A3 19991007
			JP 2000-574560	A3 19991007
			JP 2000-574686	A3 19991007
			JP 2000-574702	A3 19991007
			WO 1999-US23371	W 19991007
			JP 2005-156477	A3 20050527
			JP 2005-178654	A3 20050617

AB Animal growth regulators of the transforming growth factor β superfamily that have novel biol. activities are prepared by exchanging domains from the C-terminal active region. These new proteins may have therapeutic uses, including increased biol. activity arising from an increased efficiency of correct refolding after manufacture as inclusion bodies in bacterial hosts. In particular, domain exchange proteins derived from bone morphogenetic proteins are described.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 34 OF 41 MEDLINE ON STN DUPLICATE 19
 ACCESSION NUMBER: 2000225498 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 10760482
 TITLE: Chronic tooth pulp inflammation causes transient and persistent expression of Fos in dynorphin-rich regions of rat brainstem.
 AUTHOR: Byers M R; Chudler E H; Iadarola M J
 CORPORATE SOURCE: Department of Anesthesiology, University of Washington, Seattle, WA 98195-6540, USA.. byersm@u.washington.edu

CONTRACT NUMBER: DE05159 (United States NIDCR NIH HHS)
SOURCE: Brain research, (2000 Apr 10) Vol. 861, No. 2, pp. 191-207.
Journal code: 0045503. ISSN: 0006-8993.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 16 Jun 2000
Last Updated on STN: 16 Jun 2000
Entered Medline: 6 Jun 2000

AB We have analyzed central Fos immunoreactivity (Fos-IR) brainstems of adult rats after three clinically relevant dental injuries: filled dentin (DF) cavities that cause mild pulp injury and heal within 1-2 weeks; open pulp exposures (PX) that cause gradual pulp loss and subsequent periodontal lesions; and filled pulp exposures (PXf). By 1 week after DF cavities, no Fos-IR remained except for sites such as lateral-ventral periolivary nucleus (LVPO) that had Fos-IR in all rats including controls. PX injury induced (1) a delayed transient expression of Fos at 1-2 weeks at three loci (ipsilateral neurons in dorsomedial nucleus oralis, paratrigeminal nucleus, and trigeminal tract), (2) persistent ipsilateral Fos for at least 4 weeks after injury in dynorphin (Dyn)-rich regions (rostral lateral solitary nucleus, peribex dorsal nucleus caudalis), and (3) late Fos-IR at 2-4 weeks (bilateral superficial cervical dorsal horn, contralateral dorsal nucleus caudalis, contralateral rostral lateral solitary nucleus). Rats with PXf injury were examined at 2 weeks, and they had greater numbers and more extensive rostro-caudal distribution of Fos neurons than the PX group. One week after PX injury, Fos-IR neurons were found in regions with strong Dyn-IR central fibers. Co-expression of Dyn and Fos was found in some unusually large neurons of the ipsilateral rostral lateral solitary nucleus, trigeminal tract, and dorsal nucleus caudalis. Immunocytochemistry for the p75 low affinity neurotrophin receptor (p75NTR) or for calcitonin gene-related peptide (CGRP) showed no consistent change in trigeminal central endings in any Fos-reactive brainstem areas, despite the extensive structural and cytochemical reorganization of the peripheral endings of the dental neurons. The Fos responses of central neurons to tooth injury have some unusual temporal and spatial patterns in adult rats compared to other trigeminal injury models.

L4 ANSWER 35 OF 41 MEDLINE on STN DUPLICATE 20
ACCESSION NUMBER: 2000142040 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10678572
TITLE: Heterogeneous localizations of Trk B among individual periodontal Ruffini endings in the rat incisor.
AUTHOR: Atsumi Y; Hayashi S; Nakakura-Ohshima K; Maeda T; Kurisu K; Wakisaka S
CORPORATE SOURCE: Department of Oral Anatomy and Developmental Biology, Osaka University Faculty of Dentistry, Suita, Japan.
SOURCE: Archives of histology and cytology, (1999 Dec) Vol. 62, No. 5, pp. 435-40.
Journal code: 8806082. ISSN: 0914-9465.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20 Mar 2000
Last Updated on STN: 20 Mar 2000
Entered Medline: 9 Mar 2000

AB The present immunocytochemical study examined the localization of Trk B, a high affinity neurotrophin receptor, in the neural elements of the periodontal ligament of the rat incisor. In light microscopy, the immunoreactivity was demonstrated in dendritic profiles in the alveolar half of the periodontal ligament. Their location and morphological features indicated that they were periodontal Ruffini endings. Occasional rounded cells associated with periodontal Ruffini endings, which had immunonegative kidney-shaped nuclei, were immunoreactive; these were judged to be terminal Schwann cells. Immunoelectron microscopy revealed the heterogeneous localization of Trk B among individual Ruffini endings. Some terminal Schwann cells contained immunoreactive products for Trk B in the cytoplasm, while others did not. Similarly, a part of the Schwann sheaths covering the axon terminals showed Trk B immunoreactivity. Most axon terminals associated with periodontal Ruffini endings were immunopositive for Trk B, though a few of them were immunonegative. The ordinary Schwann cells did not contain Trk B immunoreactive products. These findings imply that Trk B is required for the maintenance of periodontal Ruffini endings. The different expression pattern of Trk B suggests that neuronal and glial elements comprising individual periodontal Ruffini endings are subject to heterogeneous conditions with regard to the requirement of Trk B.

L4 ANSWER 36 OF 41 MEDLINE on STN DUPLICATE 21
 ACCESSION NUMBER: 1999443225 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 10515202
 TITLE: Immunolocalization of Bone Morphogenetic Protein-2 and -3 and Osteogenic Protein-1 during murine tooth root morphogenesis and in other craniofacial structures.
 AUTHOR: Thomadakis G; Ramoshebi L N; Crooks J; Rueger D C; Ripamonti U
 CORPORATE SOURCE: Bone Research Laboratory, Medical Research Council/University of the Witwatersrand, Medical School, Johannesburg, South Africa.
 SOURCE: European journal of oral sciences, (1999 Oct) Vol. 107, No. 5, pp. 368-77.
 Journal code: 9504563. ISSN: 0909-8836.
 PUB. COUNTRY: Denmark
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Dental Journals; Priority Journals; Space Life Sciences
 ENTRY MONTH: 199912
 ENTRY DATE: Entered STN: 13 Jan 2000
 Last Updated on STN: 13 Jan 2000
 Entered Medline: 20 Dec 1999

AB The distribution of Bone Morphogenetic Protein-2, and -3 (BMP-2 and BMP-3) and Osteogenic Protein-1 (OP-1, also known as BMP-7) during root morphogenesis and in other craniofacial structures was examined in sections of 12- to 18-d-old mouse heads using polyclonal and monoclonal antibodies. BMP-3 and OP-1 were localized in alveolar bone, cementum, and periodontal ligament, whereas BMP-2 was only localized in the alveolar bone of periodontium. All three BMPs were localized in predentine, dentine, odontoblasts, osteoblasts, osteocytes, osteoid, cartilage, chondrocytes and spiral limbus. BMP-2 and OP-1 were also localized in spiral ligament and interdental cells of the cochlea, whilst BMP-3 was restricted to the spiral ganglion. BMP-3 was also localized in ducts of submandibular and sublingual salivary glands, acini of the lacrimal gland, Purkinje cells in the cerebellum, nerve fibres of the cerebellum and brain, afferent cells of the dorsal root ganglia, inferior alveolar nerve, and peripheral processes of the vestibulocochlear nerve. OP-1 was also localized in hair and whisker follicles, sclera of the eye

and in ameloblasts. The demonstration of BMP-3 in the nervous system suggests that this protein may be neurotrophic during development and maintenance of the nervous system. The composite expression of BMPs/OPs during periodontal tissue morphogenesis suggests that optimal therapeutic regeneration may entail the combined use of different BMPs/OPs.

L4 ANSWER 37 OF 41 MEDLINE on STN DUPLICATE 22
 ACCESSION NUMBER: 1998020689 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9382710
 TITLE: Expression of TrkB-like immunoreactivity in non-neural cells of rat periodontal ligament.
 AUTHOR: Ochi K; Saito I; Hanada K; Maeda T
 CORPORATE SOURCE: Department of Orthodontics, Niigata University School of Dentistry, Japan.
 SOURCE: Archives of oral biology, (1997 Jun) Vol. 42, No. 6, pp. 455-64.
 Journal code: 0116711. ISSN: 0003-9969.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Dental Journals; Priority Journals
 ENTRY MONTH: 199711
 ENTRY DATE: Entered STN: 24 Dec 1997
 Last Updated on STN: 22 Mar 2003
 Entered Medline: 10 Nov 1997

AB The Trk family, a group of high-affinity neurotrophin receptors, is divided into three subtypes, TrkA, TrkB and TrkC. These were originally found in neural elements, and are involved in neural development, maintenance and survival. Recent studies have shown that non-neural cells in vitro also express mRNA encoding some neurotrophin receptors. In our preliminary study, TrkB-like immunoreactivity (LI) was found in the various non-neural cells in the rat periodontal ligament. The present study was undertaken to clarify which cell types express Trk-LI, in particular two types of TrkB-LI, in the periodontal ligament of mature rats, using an immunocytochemical technique with polyclonal antibodies. Intense full-length TrkB-LI was clearly recognized in non-neural cells such as fibroblasts, osteoclasts, odontoclasts and cementoblasts as well as in neural elements. Relatively large cells with many cytoplasmic processes were also frequently immunopositive for full-length TrkB. Immunocytochemistry for TrkB[TK-], a truncated type, also demonstrated a similar immunostaining pattern to that of full-length TrkB in non-neural periodontal cells, and intense positive reactions in endothelial cells. Some non-neural cells were positive for TrkA and TrkC. These findings suggest that neurotrophic factors, the ligands of the Trk family, have certain effects on the proliferation and/or differentiation of non-neural cells, as well as on their neurotrophic functions.

L4 ANSWER 38 OF 41 MEDLINE on STN DUPLICATE 23
 ACCESSION NUMBER: 1997412016 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9268129
 TITLE: Dental innervation and CGRP in adult p75-deficient mice.
 AUTHOR: Sarraf S; Lee K F; Byers M R
 CORPORATE SOURCE: Department of Endodontics, University of Washington, Seattle 98195-6540, USA.
 CONTRACT NUMBER: DE05159 (United States NIDCR NIH HHS)
 DE11466 (United States NIDCR NIH HHS)
 SOURCE: The Journal of comparative neurology, (1997 Aug 25) Vol. 385, No. 2, pp. 297-308.

JOURNAL CODE: 0406041. ISSN: 0021-9967.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 5 Nov 1997
Last Updated on STN: 3 Mar 2000
Entered Medline: 21 Oct 1997

AB Adult dental tissues have unusual neurotrophin biology. Pulpal fibroblasts express nerve growth factor (NGF) and the low-affinity p75 neurotrophin receptor, their sensory nerve fibers express p75 and trk A, and pulpal sympathetic fibers lack p75. Following tooth injury, there is increased pulpal NGF, sprouting of sensory nerve endings, and increased immunoreactivity for the sensory neuropeptide calcitonin gene-related peptide (CGRP). In the present study, we have analyzed tooth structure and innervation of pulp and periodontal ligament in young (6-8 weeks, 3 months) and older (5-12 months) adult mice carrying a null mutation in the p75 gene and compared the results with those of age-matched wild-type controls. Our hypotheses were that tooth structure would be abnormal and that pulpal innervation would be greatly reduced because it consists primarily of nociceptive fibers that have been found to be severely depleted in skin of p75(-/-) mice. Tissues were fixed, X-rayed for gross dental morphology, decalcified, and analyzed for immunoreactivity for CGRP and for a general nerve marker, protein gene product 9.5. Radiographs showed worn-down molar crowns in p75-deficient mice. Light microscopy confirmed the accelerated molar wear and showed intense CGRP immunoreactivity in pulp nerve endings of mutant mice, compared with a gradual decrease in CGRP intensity in controls during normal aging. The CGRP intensity in 5-12-month-old pairs of mice was threefold greater in the mutants ($P < 0.03$), and in younger mice the mutant always had more CGRP than its matched control. The innervation of molar ligament in all p75-deficient mice was similar to that of controls except there was nerve sprouting near bone loss in mutants. The incisors of mutant mice did not have unusual wear and their pulpal CGRP immunoreactivity remained normal, but their periodontal ligament had fewer thin branched nerve endings at all ages. Thus, most innervation of teeth and their supporting tissues developed normally, and the only neural changes in p75(-/-) mutant mice were the reduction of incisor ligament sensory receptors and increased molar CGRP. Sensory nerves in teeth gradually lose neuropeptide intensity during aging, but that did not happen in the mutant mice, suggesting that the accelerated molar wear stimulated persistent high levels of CGRP.

L4 ANSWER 39 OF 41 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:494607 BIOSIS
DOCUMENT NUMBER: PREV199699216963
TITLE: NT3, NT4 and GDNF in tooth development.
AUTHOR(S): Nosrat, C. A. [Reprint author]; Fried, K.; Lindquist, E.; Lindskog, S.; Olson, L.
CORPORATE SOURCE: Dep. Oral Diagnostics, Karolinska Inst., Stockholm, Sweden
SOURCE: Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 302.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience, Washington, D.C., USA. November 16-21, 1996.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)

LANGUAGE: English
ENTRY DATE: Entered STN: 4 Nov 1996
Last Updated on STN: 4 Nov 1996

L4 ANSWER 40 OF 41 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 1990:441829 BIOSIS
DOCUMENT NUMBER: PREV199039089690; BR39:89690
TITLE: NEUROTROPHIC INTERACTIONS IN PERIODONTAL DISEASE.
AUTHOR(S): BARR B K [reprint author]; KELLY J P
CORPORATE SOURCE: DEP PERIODONTICS ANAT, COLUMBIA UNIV, NEW YORK, NY, USA
SOURCE: Journal of Dental Research, (1990) Vol. 69, No. SPEC. ISSUE MAR, pp. 191.
Meeting Info.: 68TH GENERAL SESSION OF THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH AND THE 19TH ANNUAL SESSION OF THE AMERICAN ASSOCIATION FOR DENTAL RESEARCH, CINCINNATI, OHIO, USA, MARCH 7-11, 1990. J DENT RES. CODEN: JDREAF. ISSN: 0022-0345.

DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 30 Sep 1990
Last Updated on STN: 1 Oct 1990

L4 ANSWER 41 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1934:55671 CAPLUS
DOCUMENT NUMBER: 28:55671
ORIGINAL REFERENCE NO.: 28:6790h-i,6791a
TITLE: Diet and the nerve supply to the dental tissues
AUTHOR(S): Mellanby, Mary; King, J. D.
SOURCE: British Dental Journal (1934), 56, 538-49
CODEN: BDJOAJ; ISSN: 0007-0610

DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Confirmation has been obtained that vitamin A deficiency results in epithelial hyperplasia, gingivitis and periodontal disease. With an adequate supply of vitamin A or carotene these tissues generally remain healthy and normal. The degeneration of the maxillary and mandibular divisions of the trigeminal nerve as well as of the origin cells of the latter observed in the animals fed on vitamin-A-deficient diets was prevented by the addition of vitamin A or carotene to such diets. Expts. are in progress to ascertain the correlation between the epithelial and nervous lesions. Loss of neurotrophic control may be partly responsible for pyorrhea and other diseases of the periodontal tissues and even of dental caries.

=> FIL STNGUIDE

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CAS SUBSCRIBER PRICE	-13.12	-13.12

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L1 87167 SEA FILE=MFE SPE=ON ABB=ON PLU=ON PERIODONTAL
L2 100 SEA FILE=MFE SPE=ON ABB=ON PLU=ON L1 AND NEUROTROPH?
L3 10 SEA FILE=MFE SPE=ON ABB=ON PLU=ON L2 AND (IMPLANT OR
TRANSPLANT)
L4 41 DUP REM L2 (59 DUPLICATES REMOVED)
L5 7 DUP REM L3 (3 DUPLICATES REMOVED)
DIS IBIB ABS L5 1-7
DIS IBIB ABS L4 1-41

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	ENTRY	SESSION
FULL ESTIMATED COST	0.84	91.15
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-13.12

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